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ORIGINAL ARTICLES.

THE ANATOMICAL EXPLANATION OF THE PARALYSIS OF THE LEFT RECURRENT LARYNGEAL NERVE FOUND IN CERTAIN CASES OF MITRAL STENOSIS.[1]

By George Fetterolf, A.B., M.D.,
DEMONSTRATOR OF ANATOMY IN THE UNIVERSITY OF PENNSYLVANIA; LARYNGOLOGIST TO THE WHITE HAVEN SANATORIUM AND THE HENRY PHIPPS INSTITUTE, PHILADELPHIA; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA,

AND

George William Norris, A.B., M.D.,
ASSOCIATE IN MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA; VISITING PHYSICIAN TO THE EPISCOPAL HOSPITAL; ASSISTANT VISITING PHYSICIAN TO THE PHILADELPHIA GENERAL AND THE UNIVERSITY HOSPITALS; PHYSICIAN TO THE OUT-PATIENT DEPARTMENT OF THE PENNSYLVANIA HOSPITAL; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.

(From the Laboratory of Anatomy of the University of Pennsylvania, Philadelphia.)

During the past thirteen years there have been reported 37 cases of mitral stenosis with associated interference with or abolition of the movements of the left vocal cord. Study of the notes of these cases which came to autopsy shows so much variation in the structures which are described as causing pressure on the left recurrent laryngeal nerve that the present study was undertaken with the idea of attempting to clear up the discrepancies. Our work has consisted of an analysis of all the cases reported up to the present time, and of a careful examination of sections and dissections of hardened thoraces. Observations made in soft bodies are of questionable value, because the opening of the chest allows of so much collapse and the manipulations of dissection cause such a degree of distortion that normal relations are altered to a most astonishing degree. We therefore used only cadavers with everything hardened *in situ* by means of an arterial injection of formaldehyde solution, in the endeavor to keep the thoracic and especially the mediastinal viscera as nearly as possible as they are *in vivo*.

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[1] Read at a meeting of the College of Physicians of Philadelphia, March 1, 1911.

HISTORICAL. The association of paralysis of the left recurrent laryngeal nerve with mitral stenosis was first described by Ortner, in 1897, as occurring in two cases which had come under his observation. He ascribed the paralysis to compression of the nerve between the arch of the aorta and the distended left auricle. The nerve in his cases was discolored, compressed, and ribbonlike. In the following year Herrick recorded a case in which, in addition to a dilated left auricle, chronic adhesive pericarditis was found, the nerve being embedded in a mass of cicatricial tissue as well as compressed by the auricle.

In 1901 Kraus reported a case of left recurrent paralysis associated with mitral stenosis. He took exception to the anatomy of Ortner's explanation, and described the mechanism in his case quite differently, taking the ground that the laryngeal paralysis, although associated with marked left auricular dilatation, was indirectly due to hypertrophy of the right ventricle. This condition, by altering the position of the heart as it lay on the diaphragm, was assumed to alter the relationship between the pulmonary artery, the aortic arch, and the aortic ligament in such a way as to cause the ligament to drag upon the recurrent laryngeal nerve. In his case the aortic ligament is stated to have run more horizontally than is normal, so that the left recurrent laryngeal nerve did not course laterally from the aortic ligament around the aortic arch, but crossed the latter.

At the point at which the nerve crossed the aortic ligament it was constricted and discolored.

In v. Schroetter's case the nerve is reported as having been compressed between a patulous ductus arteriosus and the aortic arch, and in Frischauer's case, between the left pulmonary artery and the aorta, the former being pushed upward by the dilated left auricle. Hofbauer described the compression as occurring between the dilated pulmonary artery and the aorta, while in Bonardi's and in two of Osler's cases we are again told that the left auricle impinged directly upon the aorta. In Osler's third case there was no autopsy.

In Gantz's case the nerve is stated to have been pressed upon by enlarged peribronchial and peritracheal glands. Despite the coexistence of a severe pneumonia and pleuritis, he attributes this glandular enlargement to stasis produced by a weak heart. No similar explanation is to be found in the literature of the subject. In Mead's case the compression was presumably exerted by the greatly dilated pulmonary artery, although a patulous ductus arteriosus was also found.

Little of clinical interest is reported concerning the laryngeal symptoms, although in some of the cases it was noted that the hoarseness was more marked in certain positions. Thus, Hofbauer's patient was less hoarse in the dorsal and right lateral postures, while Prota's[2] was more hoarse when the head was rotated to the left.

The paralysis may apparently be bilateral. In seven of Quadrone's eight cases both cords were paretic. In explanation of this he assumes that the brachiocephalic and subclavian arteries may sustain the brunt of the cardiac displacement. It is on this basis that the inequality of the radial pulses sometimes met with in cases of mitral disease has been explained (pressure of the auricle upon the aortic origin of the left subclavian artery). Thus, Massei[3] and Perotta hold that right-sided recurrent paralysis in mitral stenosis is never found as an isolated or primary lesion, but only as an exaggeration of those conditions which brought about the left recurrent paralysis. As a matter of fact, the entire situation, especially when the right recurrent is concerned, is so clouded that it is eminently desirable that future autopsies should be made with greater care, or, as Kraus has suggested, not made as routine autopsies, but as frozen sections. None of the cases in which the right nerve was implicated have come to autopsy.

ANATOMIC CONSIDERATIONS. The left recurrent nerve springs from the vagus as the latter is passing down the sinistro-anterior aspect of the horizontal part of the aortic arch (Fig. 1). It hugs the aorta closely and passes under the arch either at the point at which the ligamentum arteriosum joins the latter vessel or slightly anterior to this position (Fig. 1). Reaching the dextro-posterior side of the arch, it ascends, its further course having no bearing on the clinical condition under discussion.

The bifurcation of the pulmonary aorta takes place at the lower inner margin of the left bronchus, about 2.5 cm. from the bifurcation of the trachea. The angle lies behind the left margin of the ascending part of the aortic arch and above and in front of the root of the left upper pulmonary vein (Figs. 2 and 3). The right branch passes horizontally to the right under the aortic arch and above the left auricle. It does not enter the present problem. The left branch curves over the left superior pulmonary vein and the left bronchus, both of which it indents, and passes outward and markedly backward, forming an angle with the right branch of about 100 degrees in the horizontal plane. Above, and 4 mm. away from it is the beginning of the descending part of the aortic arch, to which it is connected by the ligamentum arteriosum (Fig. 1). The descending part of the arch continues down in contact with the posterior mesial surface of the left pulmonary artery. Below the left pulmonary artery lie the left superior pulmonary vein and the left bronchus (Figs. 2 and 3).

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[2] "Su due casi di emiplegia laryngea con singulare disturbo disfonico." Atti del Terzo Congresso d. Soc. Italiana di Larengologia, 1899, p. 230. (No pathological findings in thorax in either case clinically. No autopsies.)

[3] Quoted, Boinet, l. c.

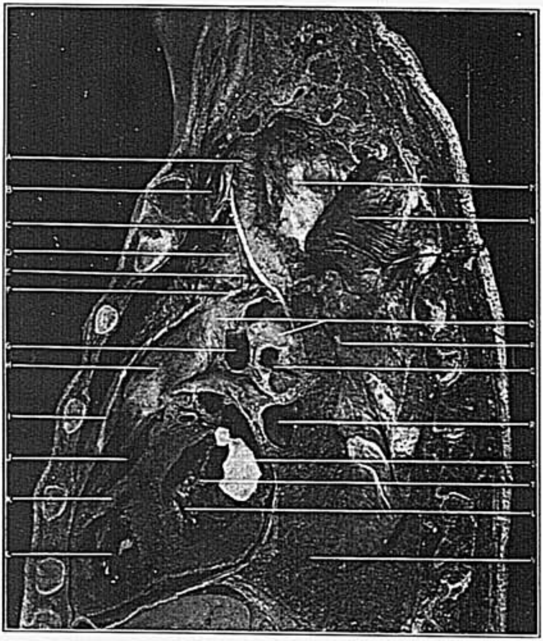

FIG. 1.—Sagittal section 3 cm. to the left of the midsternal line, viewed from the left: *A*, left subclavian artery; *B*, retracted pleura; *C*, left vagus nerve; *D*, arch of aorta; *E*, left recurrent laryngeal nerve; *F*, ligamentum arteriosum; *G*, left superior pulmonary vein; *H*, pulmonary aorta; *I*, reflected pericardium; *J*, right ventricle; *K*, moderator band; *L*, right ventricle; *M*, apical mediastinal pleura; *N*, *P*, retracted upper lobe of left lung; *O*, left pulmonary artery; *Q*, left bronchus; *R*, left inferior pulmonary vein; *S*, left auricle; *T*, mitral leaflet; *U*, left ventricle; *V*, cut surface of left lower lobe.

The appendix of the left auricle is curled around the root of the pulmonary aorta. It projects upward in the angle between the left pulmonary artery and the left superior pulmonary vein (Fig. 4).

The ligamentum arteriosum (the obliterated ductus arteriosus) is a fibrous cord, about 3 mm. thick and 2 cm. long. We have found that it lies almost exactly in the antero-posterior plane of the body and ascends but slightly as it passes backward from the left pulmonary artery to the aorta (Fig. 1). In or slightly anterior to the obtuse angle formed at its junction with the latter vessel lies the recurrent laryngeal nerve.

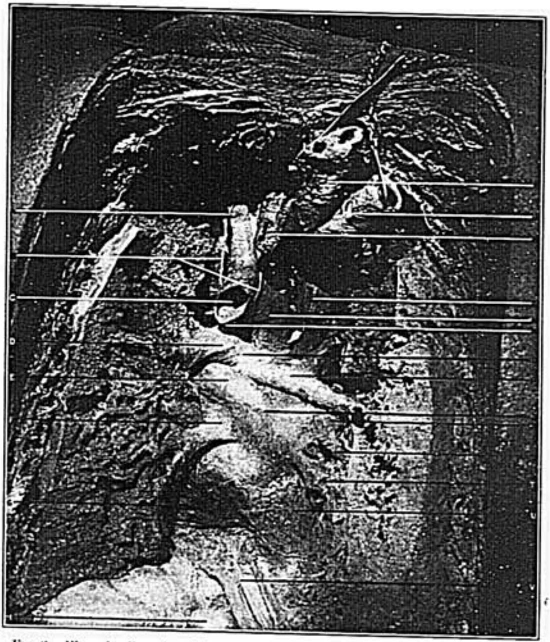

FIG. 2.—View of a dissection of the posterior mediastinum from behind: *A*, left subclavian artery; *B*, left recurrent laryngeal nerve; *C*, aorta; *D*, left pulmonary artery; *E*, left superior pulmonary vein; *F*, left inferior pulmonary vein; *G*, left ventricle; *H*, aorta (reflected); *I*, left bronchus (reflected); *J*, right bronchus (reflected); *K*, cut end of esophagus; *L*, superior vena cava; *M*, descending part of aortic arch; *N*, ligamentum arteriosum; *O*, angle of bifurcation of pulmonary aorta; *P*, right pulmonary artery; *Q*, left auricle; *R*, right superior pulmonary vein; *S*, right middle pulmonary vein; *T*, right inferior pulmonary vein; *U*, inferior vena cava; *V*, esophagus (reflected).

In effecting pressure on the nerve there are two conditions at work

—increase in size and alteration of position, both dependent upon the narrowing of the mitral orifice. Changes in size, viz., enlargement, involve in sequence the left auricle and appendix, the pulmonary veins, and the pulmonary arteries. The obstruction to the blood current in the mitral orifice results, first, in a dilatation of the left auricle and its auricular appendix. Rise of pressure in this chamber is followed by the same condition in the pulmonary veins, which results in their overfilling and distention. This in time dams back the blood in the lungs and tends to cause its stagnation in the pulmonary artery and in the right heart. In consequence there is always present a dilatation of the left auricle and of the pulmonary

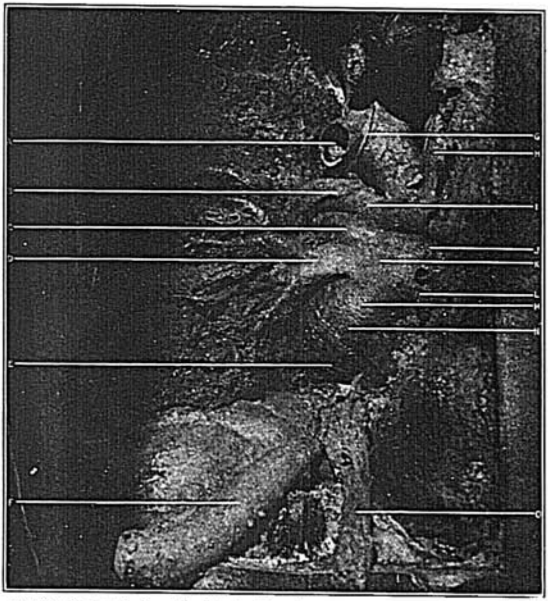

FIG. 3.—Oblique view from the right of a dissection of the mediastinal viscera from behind: *A*, aorta; *B*, left pulmonary artery; *C*, left superior pulmonary vein; *D*, left inferior pulmonary vein; *E*, left ventricle; *F*, thoracic aorta (reflected); *G*, left recurrent laryngeal nerve; *H*, superior vena cava; *I*, angle of bifurcation of pulmonary aorta; *J*, root of right superior pulmonary vein; *K*, *M*, left auricle; *L*, right inferior pulmonary vein; *N*, coronary sinus; *O*, esophagus (reflected).

arteries and veins, which gives rise to a crowding of the mediastinal structures at the base of the heart.

Changes in position are mainly due to the distention of the auricle, although this can be aided materially by enlargement of the right heart, particularly of the ventricle. Some remarkable instances of left auricular dilatation have been recorded. According to Witwicki,[4] the left auricle is the most distensible of all the heart chambers, and Samuelson[5] has stated that occlusion of the left coronary artery causes a flaccid dilatation of the auricular walls, which may assume the appearance of a distended bladder. Minkowski[6] removed a heart at autopsy which contained about 4 liters of blood, of which nearly 3 were found in the left auricle. This specimen was obtained from the body of a man, aged thirty-two years, who had suffered from double mitral and tricuspid lesions and in whom a diagnosis of mitral stenosis had been made fourteen years before his death.

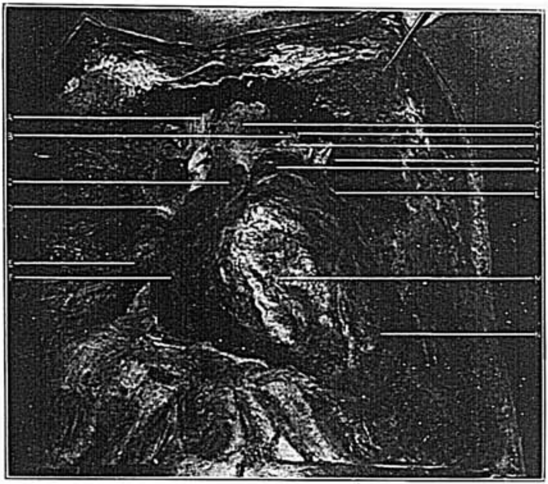

FIG. 4.—View of a dissection of the mediastinal viscera from in front: *A*, anterior vena cava; *B*, ligamentum arteriosum; *C*, right auricular appendix; *D*, right superior pulmonary vein; *E*, right inferior pulmonary vein; *F*, right auricle; *G*, arch of aorta; *H*, left recurrent laryngeal nerve; *I*, left pulmonary artery; *J*, left superior pulmonary vein; *K*, pulmonary aorta; *L*, left auricular appendix; *M*, right ventricle; *N*, left ventricle.

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[4] Zeit. f. klin. Med., 1895, xxvii, 321.

[6] Münch. med. Woch., 1901, p. 182.

[5] Soc. Anat., Paris, vol. ii.

Despite this enormous dilatation the patient had been able to walk about until but shortly before his demise. The auricular walls were thin and membranous. In the case reported by Briquet[7] the left auricular capacity was 650 c.c. (the normal average being 55 c.c.), or what is practically equal to the capacity of a whole normal heart. Müller[8] has recorded an instance in which the left auricle was larger than a child's head, this occurring in a case in which the heart weighed 620 grams, the patient weighing only 65 kilograms. (Under normal circumstances a patient of this weight should have a heart weighing about 300 grams). Toledano[9] reported a case in which the auricle measured 12 by 11 cm. Sansom has described the auricular wall as being attenuated to the thinness of a visiting card. Owen and Fenton[10] have reported a left auricle with a capacity of 900 c.c., a distention so great that aspiration was performed in the belief that a pericardial effusion was present.

It is well known that dilatation of the pulmonary artery is often encountered in long-standing cases of mitral stenosis, and not infrequently the artery undergoes considerable arteriosclerotic change. In these cases there is an area of dulness at the upper left margin of the sternum, sometimes sensitive to pressure or percussion, as well as enlargement of the right heart. Dmitrenko, in his case of pressure paralysis, succeeded in making graphic records of the pulmonary pulsation.

The left auricular cavity can expand readily in but one direction, viz., upward (Figs. 2 and 3); posteriorly, it rests against the aorta and esophagus; anteriorly, it is limited by the right auricle and the left ventricle, and below by the right auricle and the liver. In expansion, the auricular appendix would probably be the first part affected, as it lie, free and unadherent to anything. Should it become dilated it would press upward and backward, thrusting the left pulmonary artery against the aorta, the left upper pulmonary vein against the left pulmonary artery, and forcing the distal portion of the latter against the aorta. Later, when the atrium or main cavity of the auricle dilates, the proximal part of the left pulmonary artery is jammed upward and backward, mainly by the root of the left upper pulmonary vein. The nerve is thus squeezed between the artery on the one hand and the aortic arch or the ligamentum arteriosum on the other. Frischauer and Hofbauer are the only ones who report finding this post mortem.

COMMENT ON PREVIOUS EXPLANATIONS. The foregoing facts should make us extremely cautious in accepting the reports of non-autopsied cases as examples of recurrent paralysis due to auricular pressure in the course of mitral stenosis. It should be borne in mind that the first examples of this condition were first reported fully thirty-five years after paralysis of the recurrent laryngeal nerve had first been described by Tuerck and Gerhardt (1862). Furthermore, the details of the cases reported by Koellreuter, in which the paralysis was in all probability due to a mediastinal neoplasm, and by Gantz and ourselves, in which glandular enlargement was the etiologic factor, clearly show how closely the picture produced by mitral stenosis may be simulated by other causes and conditions.

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[7] "De l'état du cœur gauche dans les lesions mitrales." Thèse de Paris, 1890.

[8] Zeit. f. klin. Med., 1905, lvi, 520

[9] Soc. Anat., Paris, 1875.

[10] Clinical Society, London, May 24, 1901.

The junior author has for the last six years been especially on the lookout for this conjunction of pathological conditions, and among many hundreds of cardiac patients seen during this time has met with only one case in which recurrent paralysis appeared to be due to mitral disease. There was, however, no autopsy, and the few notes which were made have been lost.

How easily one might be led into attributing left recurrent laryngeal paralysis to mitral stenosis is also illustrated in the following case, which occurred in the service of Dr. F. H. Klaer, in which a diagnosis of mitral stenosis was readily made and in which aortic aneurysm was suspected but later abandoned as the result of the *x*-ray examination, which disclosed the presence of enlarged bronchial lymph nodes.

W. A. H., male, aged twenty-nine years, a clerk by occupation, presented himself at the medical dispensary of the University Hospital on account of loss of weight (18 pounds), indigestion, and hoarseness when in the prone position. This latter symptom, which had been present for four weeks, was sometimes relieved by belching. He had no symptoms indicative of cardiac weakness. He had marked enlargement of the axillary lymph nodes and physical signs indicative of an infiltration of the left pulmonary apex.

Heart. Dulness extends from upper border of the third rib just below the right sternal border to one finger's breadth outside the midclavicular line. There is a marked presystolic thrill. On auscultation at the apex a rumbling presystolic sound is heard, ending in a loud booming systolic sound. The pulmonic second sound is greatly accentuated. The temporal arteries are sclerotic, the radial pulse has a small volume, that in the left being smaller than that on the right.

On further examination, an area of dulness above the heart and to the left of the sternum was discovered, which suggested the existence of an aortic aneurysm.

Laryngeal Examination (Dr. Singer). Complete paralysis of the left vocal cord, without any other signs of local abnormality.

X-ray Examination (Dr. Pancoast). The heart is enlarged after the manner indicated by the physical examination. The bronchial lymph nodes are enlarged.

Later on the patient developed cough when in the recumbent posture. The heart became more dilated, and the presystolic murmur less loud. About this time he became confined to bed, his symptoms having progressively increased in severity. His private physician and a consultant, on being called to see him, pronounced his case one of thoracic aneurysm with cardiac enlargement. Death occurred as the result of edema, dyspnœa, etc. No autopsy was held.

Of all internal maladies, disease of the aorta is most frequently the cause of left recurrent laryngeal paralysis Among 69 such cases Syllaba found 17, while Guder and Dufour encountered 8 cases among 79 (2 of complete paralysis and 6 of paresis) due to this cause. In 1 of Ortner's and in 1 of Osler's cases aortic aneurysm had been diagnosticated, and not unnaturally, since disease of the aorta and mitral stenosis may have many symptoms in common, among which inequality of the radial pulses and anisocoria,[11] cyanosis, cough, dyspnœa, aphonia, and pulsation in the second left intercostal space may be especially mentioned. Indeed, Boinet has suggested that periaortitis may cause a recurrent paralysis by direct extension without any compression whatever. It is also possible that antecedent infections, especially diphtheria, may give rise to the vocal paralysis. Chronic adhesive pericarditis, which was found in 3 of the reported cases, might readily account for the nerve paralysis. In Herrick's case the left recurrent laryngeal nerve was imbedded in a mass of cicatricial tissue.

The cases reported by Gantz and by Palasse were in all probability also the result of glandular pressure, a process in which it is incredible that the heart should have played any part. Pulmonary tuberculosis may cause recurrent paralysis either by direct glandular pressure—a gland is normally found between the bronchus anteriorly and the nerve posteriorly—or as the result of pleuritis, or through involvement of the pericardium or pulmonary consolidation. The knowledge of these facts makes one distinctly skeptical of the accuracy of Ceraulo's observations of twenty cases supposedly due to mitral stenosis. Quadrone reports eight cases in which paresis or paralysis of the cords occurred in cases of mitral disease, without any changes in the laryngeal mucosa, without any demonstrable cause other than the heart lesion. Three cases were examined with the *x*-rays. The reports of Syllaba, Alexander, Gavello, Quadrone, Perotta, Sheldon, Guder and Dufour, Pallasse, Pal, Ceraulo, Mead, Dmitrenko, and Koellreuter being unsubstantiated by autopsies, leave us in much uncertainty as to the actual mechanism of the compression, though they seem sufficient to emphasize the relationship of the two pathological conditions.

The observations of Ortner to the effect that the pressure was exerted by the left auricle against the left bronchus in one case and against the aorta in the other, would seem to be faulty. To have the auricle press upon the aorta would necessitate that the pulmo-

[11] Harris, Harvein Society, London, 1903.

nary artery unguard the nerve by being thrust not only backward but downward and away from the aorta in a manner and to a degree that is practically inconceivable. As regards pressure upon the trachea by the auricle, it is anatomically impossible to have the recurrent laryngeal nipped between the left auricle and the bronchus. This statement is made despite the fact that both King and Friederich[12] have reported compression of the left bronchus by a dilated left auricle, an entirely different matter, and occurring below the lowest part of the recurrent laryngeal loop. These errors, like many others, tend to support the belief that the ordinary method of performing autopsies will not reveal the minutiæ of interrelational thoracic anatomy. Only by having the body hardened before it is studied can such intimate details be determined with exact definiteness.

Kraus' explanation of the mechanism at work has more to commend it, although he is in error in regard to some of his anatomical relations. In addition to marked dilatation of the left auricle, he ascribes some of the trouble to a hypertrophied right ventricle, with consequent alteration of the relations at the aortic arch. He states that in his case the aortic ligament, instead of running upward and to the left, ran more horizontally. As a matter of fact, the abnormal course described by Kraus is the normal one. However, the ligament may have been more horizontal than usual, and this was probably caused by a pushing up of the pulmonary artery and not by a pulling down of the aorta.

In this connection it should be stated that the possibility of traction on the aorta by the pulmonary artery through the medium of the ligamentum arteriosum can hardly be looked upon seriously. The ligament runs antero-posteriorly and slightly upward, and before any downward traction could begin to be exerted the pulmonary artery would have to descend from 1 to 2 cm. It does seem a bit fanciful to assume that a structure but 2 to 3 mm. in diameter could pull downward the arch, supported as the latter is at both ends, and held up not only by the great vessels which arise from its convexity, but also by the attachment of the deep cervical fascia to the pericardium. These would certainly counteract any possible pull exerted by the ductus, to say nothing of the aid afforded by the areolar adhesions of the aorta to the adjacent mediastinal structures.

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[12] Quoted Huchard, Maladies du Cœur, 1905, iii, 522.

TABULATION OF REPORTED CASES.

Observer.	Age.	Sex.	Cardiac lesion.	Autopsy findings: remarks.
Ortner, Wien. klin. Woch., 1897, No. 33	17	M	Double mitral and tricuspid; obliterative pericarditis	Nerve compressed between auricle and left bronchus; nerve discolored
	34	F	Double mitral and aortic; tricuspid insufficiency	Nerve compressed between left auricle and aorta; nerve flattened, constricted in spots and discolored
Herrick, Chicago Med. Recorder, 1898	38	M	Mitral stenosis; obliterative pericarditis and pleuritis	Nerve embedded in cicatricial tissue "and wedged between aorta and enormously enlarged left auricle that had forced its way in between the aorta and pulmonary artery considerably distorting the normal anatomical relations of the parts;" nerve flattened, narrow and attenuated and on microscopic examination was found degenerated; auricles greatly dilated.
Kraus, Verhandl. d. XIX. Kongr. f. inn. Med., 1901, p. 607	21	F	Double mitral; tricuspid insufficiency; slight aortic stenosis	Heart displaced by hypertrophied right ventricle, altering relationship of aorta, aortic ligament and pulmonary artery, with resulting traction on the nerve.
v. Schrötter, Zeit. f. klin. Med., 1901, p. 160	15	F	Double mitral, tricuspid insufficiency; patent ductus arteriosus; dilatation of pulmonary artery	Nerve compressed between origin of ductus arteriosus and aorta; constricted and discolored; trunk of pulmonary artery larger than ascending aorta, its two branches larger than normal; ductus arteriosus as large as the latter.
Hofbauer, Wien. klin. Woch., Oct. 9, 1902	32	M	Mitral stenosis	X-ray findings identical with those of Firschauer; no autopsy.
Syballa, Sem. Méd., 1903, p. 44	47	M	Mitral stenosis	No autopsy.
Sheldon, Medical Rec., Nov., 1904	38	F	Mitral stenosis	No autopsy; vocal cord paralysis appeared and disappeared with broken compensation and improvement respectively.
Quadrone, "le paralisi delle corde vocali nei vizi mitralici," Scritti Medici in onore di C. Bozzolo, 1904, p. 515	..	F	Mitral stenosis and insufficiency	No autopsy notes; bilateral paresis; nerve histologically normal.
	..	F	Mitral stenosis	No autopsy; bilateral paresis.
	..	F	Mitral stenosis and insufficiency	No autopsy; bilateral paresis.
	..	F	Mitral stenosis and insufficiency	No autopsy; bilateral paresis.
	..	M	Mitral stenosis and insufficiency	No autopsy; bilateral paresis.
	..	F	Mitral stenosis	No autopsy; bilateral paresis.
	15	F	Mitral stenosis	No autopsy.
	..	F	Mitral stenosis and insufficiency	No autopsy; left rec. paralysis.
Alexander, Berlin klin. Woch., 1904, p. 135	50	F	Mitral stenosis	X-ray findings identical with those of Firschauer; no autopsy.
Firschauer, Wien. klin. Woch., 1905, p. 1383	30	F	Double mitral; tricuspid insufficiency	Nerve compressed between left pulmonary artery and aorta by forward and upward pressure of dilated left auricle and pulmonary vein.
Trétrop, Bull. de la Soc. belge d'otolaryngologie, 1905, p. 180	49	M	Mitral insufficiency	No autopsy; paralysis appeared and disappeared as patient got worse or better.
Hofbauer, Wien. med. Gesellsch., Nov. 14, 1905	30	M	Mitral stenosis	Nerve compressed between left auricular appendix and aorta, the former being pushed upward between the pulmonary artery and aorta.
Pal, Sem. Méd., 1905	..	..	....	No autopsy; brief verbal report.
Bonardi, Gazz. Med. Italian, 1906, p. 41	40	F	Double mitral tricuspid insufficiency; pericarditis	Nerve compressed between aorta and enormously dilated left auricle.
Koellreuter, Monatsch. f. Ohrenheilk., 1907, p. 1	29	F	....	No autopsy; x-ray, tracheoscopic, and œsophagoscopic examinations; mediastinal neoplasm; points out fallacy of accepting non-autopsied cases.

TABULATION OF REPORTED CASES—(*Continued*).

Observer.	Age.	Sex.	Cardiac lesion.	Autopsy findings: remarks.
Zimbler, Thesè de Bale, 1907	25	F	Congenital pulmonary stenosis and insufficiency; patulous ductus arteriosus	No autopsy; *x*-ray examination similar to that in v. Schrötter's case.
Gavello, Bull. di Malattie di Orechio, Nov., 1905	19	F	Mitral stenosis.	No autopsy; *x*-ray excluded possibility of disease of the aorta.
Ceraulo, Morgagni, 1907, No. 6	..	..	....	No autopsies; claims to have seen 20 cases in mitral disease, of which two were stenosis; no details.
Perotta, Arch. Ital. di Laryngolog., 1909, p. 71	38	F	Mitral stenosis	No autopsy; tertiary syphilis.
Guder and Dufour, Rev. de Méd., 1909, p. 300	43	F	Double mitral	No autopsy; partial paralysis; *x*-ray examination.
	24	M	Double mitral	No autopsy; adhesive pericarditis?
Osler, Arch. de Mal. du Cœur, 1909, p. 74	45	F	Mitral stenosis	Nerve compressed between left auricle and aorta?
	27	F	Double mitral	No autopsy.
	48	M	Double mitral with aortic insufficiency	Nerve compressed between left auricle and aorta. It was white and sclerotic; auricle size of small fist.
Boinet, Bull. de l'Acad. de Méd., 1910, p. 211	23	F	Mitral stenosis	No autopsy; paralysis appeared and disappeared as heart action became worse or better.
	25	F	Double mitral	No autopsy; *x*-ray showed great auricular dilatation, the aorta being apparently normal.
Pallase, Lyon Médicale, 1909, p. 719	20	F	Mitral stenosis	No autopsy; pulmonary tuberculosis; mediastinal adenitis revealed by *x*-ray.
Mead, Jour. Am. Med. Assoc., 1910, lv, 2205	..	F	Patulous ductus arteriosus; aortic and mitral stenosis; coronary and aortic sclerosis	Exact mechanism not stated; compression presumably due to dilatation of pulmonary artery which was distended to twice the size of the aorta; recurrent laryngeal nerve both macroscopically and microscopically normal.
Dmitrenko, Rousskyi, Vratch, 1910, No. 1; Abs. Arch. de Mal. du Cœur, Jan., 1911, p. 48	29	M	Mitral stenosis	No autopsy; paralysis attributed to dilatation of pulmonary; artery; tracings were taken from same in the pulsating second left intercostal space; *x*-ray showed left auricular enlargement.

The obliterated ductus arteriosus is mentioned frequently in the autopsy records. It is our opinion that this structure is a factor of minor or even negligible importance, its main and perhaps only influence, aside from its mere presence, being to hold in approximation to a very slight degree the aorta and the left pulmonary artery. Even should the ductus be patulous, it does not follow that it has any intrinsic influence. We are inclined to believe that in these cases, such as v. Schrötter's and Mead's, the two factors of immediate significance are identical with those in which the ductus is obliterated, viz., the aorta and the left pulmonary artery, the patulousness of the ductus being of importance only on account of the associated dilatation of the pulmonary artery and the engorgement and hypertrophy of the right ventricle.

SUMMARY. There are now on record 11 autopsied and 26 clinically reported cases in which recurrent laryngeal paralysis was associated with and apparently the result of mitral stenosis. Among the autopsied cases the vocal paralysis was attributed to direct compression on the part of the auricle or its appendix in 7; to cardiac displacement, traction, etc., in 1; to the effects of a persistently patulous ductus arteriosus in 2; and to indirect compression acting on the pulmonary artery in 2.

CONCLUSIONS. It is our conviction, based on careful study of the anatomical relations in hardened preparations, that the indirect mechanism may be a variable one, but that when compression is accountable for the recurrent paralysis, it must always be caused by the nerve being squeezed between the left pulmonary artery and the aorta or the aortic ligament.

Anything which will dilate or force upward the left auricle, the left upper pulmonary vein, or the left pulmonary artery would tend to cause the condition.

The anatomic relations are such that direct pressure of any portion of a dilated left auricle upon the aortic arch is impossible.

When we consider the softness of all the structures involved, and the fact that the nerve is normally flattened against the aorta, not rounded, it would seem probable that its function is abolished. not from actual destruction from pressure, but from a neuritis consequent upon a degree of compression which could hardly be sufficient to actually destroy the vitality of the nerve. This, of course, can be determined only by microscopic examination of the nerve.

ARNETH'S METHOD OF BLOOD COUNTING—ITS PROGNOSTIC VALUE IN PULMONARY TUBERCULOSIS.[1]

BY C. L. MINOR, M.D.,

AND

P. H. RINGER, M.D.,

ASHEVILLE, N. C.

THE great difficulty of drawing a correct prognosis in a case of pulmonary tuberculosis is too well known to need more than a mention here.

Those most experienced in the treatment of this disease frequently fail to predict correctly the future course of a case, or to estimate with any degree of accuracy its chances of recovery. Therefore, any procedure which promises to be of assistance in drawing a prognosis demands our attention and study, and hence we feel justified in taking the time of this Association to present the conclusions we have formed from the study of the blood in a hundred cases of pulmonary tuberculosis by Arneth's method.

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[1] Read before the American Climatological Association at Old Point, Va., June, 1909.

This method, developed by its author in Leube's clinic in 1904-05, has so far not attracted much attention in this country, though two of the members, of this Association, Dr. A. C. Klebs, and Dr. Geo. E. Bushnell, U. S. A., have made valuable contributions to the subject.

The former reported to the National Association for the Study and Prevention of Tuberculosis in 1906, and expressed the hope that his report might induce further investigation on these lines; but since that time, so far as we know, only Bushnell and Trueholtz have written on the subject.

Wishing to determine the value of this method as an aid to prognosis, we have examined the blood of somewhat over one hundred cases in Dr. Minor's practice, and as a result of this work we believe it has a distinct value in enabling one to estimate the resisting power, and hence the likelihood of recovery and general prognostic outlook, of such cases, and trust it will be more generally used by the profession than has been the case in the past.

As you all know, Arneth considered that the young white cells—those with one or two nuclei—are less resistant and less able to combat an infection than the older ones with more nuclei.

Starting out with this assumption, he counted the number of nuclei in all the neutrophilic white cells, and divided them into five classes, according to the number of their nuclei. Placing these numbers in a horizontal row, beginning on the left with Class I, which contained one nucleus, and ending on the right with Class V, which contained five nuclei, he formed what he called the neutrophilic blood picture. From a count of fifteen normal individuals he formed a normal picture.

I	II	III	IV	V
5 per cent.	35 per cent.	41 per cent.	17 per cent.	2 per cent.

Studying the blood in pulmonary tuberculosis, he found, in consonance with his theory, that the normal percentage of nuclei in the different classes was lost, there being an increase of the younger cells of Classes I and II, and a lessening of the more mature ones in Classes III, IV, and V, this being more marked in proportion to the severity of the trouble—one of the worst pictures he got being a case of miliary tuberculosis:

I	II	III	IV	V
36 per cent.	56 per cent.	8 per cent.	0 per cent.	0 per cent.

And again, in the case of subacute tuberculosis:

I	II	III	IV	V
45 per cent.	52 per cent.	3 per cent.	0 per cent.	0 per cent.